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THE VENOUS SYSTEM AND ITS ROLE IN THE CIRCULATION*

For many years, the varied functions of the arterial system have been discussed and emphasized, while the venous system usually has been considered to be nothing more than a passive conductor of blood from the capillaries back to the heart. The more we learn about the veins, however, the more we realize that this concept of passive nature is far from correct. For instance, the veins are provided with a pumping mechanism to make blood flow uphill. They are the major storehouse for extra blood in the circulation. And finally, it is rapidly becoming apparent that the venous system is the major regulator of venous blood flow into the heart and consequently the regulator of cardiac output itself. Therefore, the veins obviously are far more than a mere passive set of tubes.

Transmission of Blood Through the Venous System

Blood is made to flow through the veins by pressure from the capillaries, and flow through the capillaries is caused by pressure from the arteries. That is, a "force from behind" called *vis a tergo*, makes the blood flow toward the heart. Some physiologists believe that the heart might also suck blood to a slight extent from the veins because of the elastic recoil of the ventricular walls during diastole.[1, 2] This effect is called *vis a fronte*, or "force from the front." But this sucking action is probably of very minor importance because, instead of transmitting the suction into the more peripheral veins, the veins of the chest merely collapse in the same manner that a balloon collapses when one sucks on it.[3-5]

One of the most difficult problems to overcome in the flow of blood from the lower veins to the heart is the weight of the blood itself. When a person is standing and is completely still, this weight is great enough to make the blood pressure in the foot veins about 100 mm. Hg. The pressure in the foot capillaries is even higher, and this, if it lasts long enough, can cause very severe edema. To offset the effects of hydrostatic pressure, a special mechanism, the *venous pump*, operates continuously. Situated every few centimeters along the course of the peripheral veins are valves that allow blood to flow forward but prevent backflow. Therefore, compression of one of these veins can make the blood move toward the heart but not in the other direction. Each time a person contracts a muscle, compresses the skin or even passively bends a joint, a small amount of blood is pushed toward the heart. This is the origin of the term, "venous pump."

The venous pump is so effective in pushing blood uphill that when one stands as one normally does, shifting one's weight every now and then, the pumping is sufficient to keep the pressure in the foot veins low enough so that edema will not develop. When the valves in the veins become nonfunctional, however, the well-known picture of varicose veins ensues, with protruding veins, edema, and even ulceration of the skin.

Storage of Blood in the Veins

The muscular walls of the veins have a unique characteristic called "plasticity," or "stress relaxation," that allows their volumes to change readily with only slight changes in pressure. This can be explained as follows: If a large amount of blood is forced into a vein, the veins immediately become distended and the venous pressure rises abruptly, but after a few seconds the venous wall begins to stretch, allowing the pressure to fall during the ensuing minute or more to a value not far above the original pressure.[6, 7] Later, if the extra blood is removed from the vein, the diameter of the vessel suddenly decreases and the pressure falls for a short period of time. But during the next few seconds to a few minutes, the muscular wall assumes progressively increasing tone until the pressure rises almost back to normal. Thus, the veins are capable of enlarging and contracting many fold

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and still of maintaining almost constant pressure on the inside.

This plasticity characteristic makes the veins excellent storage areas for blood. For instance, after a very large transfusion has been administered to an animal, the venous blood volume is increased very greatly while the arterial volume remains almost normal. Also, in diseases in which the blood volume becomes increased, such as in congestive heart failure, polycythemia vera, and arteriovenous fistulae, most of the increased blood volume is accommodated in the veins or in the venous organs, the liver and spleen.[8] This storage of blood occurs fairly generally throughout the venous system, though special areas such as the large venous beds of the abdomen, the veins of the lungs, and the venous plexuses beneath the skin are capable of storing extra large amounts.

Sympathetic Control of the Venous System

The veins, like most of the arteries of the body, are usually contracted by sympathetic stimulation.[9, 10] Furthermore, excitation of the sympathetic nervous system usually affects both the arteries and the veins at the same time. The most important effect on the arterial tree is to increase the arteriolar resistance while the principal effect on the venous system is to increase the pressures inside all the veins[11-13] causing them to force their stored blood into those parts of the circulation which are momentarily in need of blood.

For instance, following hemorrhage of as much as 15 to 25 per cent of the total blood volume, the veins contract, greatly decreasing their volume, while the remaining portions of the circulation still retain almost normal amounts of blood.

Regulation of Cardiac Output by the Veins

One of the most important qualities of the veins is their ability to control the flow of blood into the heart—that is, to control "venous return"—and in this way to control cardiac output itself. The veins control venous return by two different methods, one of which acts rapidly and the other very slowly. The rapid method is to increase or decrease the so-called "mean circulatory pressure," the average pressure of all the blood in the body.[14, 15] When the muscular walls of the blood vessels everywhere in the circulation contract, the pressures throughout the circulation increase, causing the mean circulatory pressure to rise. And most of this rise results from increased venous tone because about two-thirds of all the blood is in the veins.[16] Furthermore, the mean circulatory pressure is the average force pushing blood from the vessels toward the heart.[17] Therefore, the higher the mean circulatory pressure—that is, the higher the average pressure in the peripheral vessels—the more rapidly will blood flow toward the heart and consequently the greater becomes the cardiac output. Experiments have shown, for instance, that stimulation of the vascular system by injection of epinephrine increases the mean circulatory pressure an average of about 200 per cent and at the same time increases the cardiac output an average of 175 per cent. Sympathetic stimulation causes the same effect. In other words, an increase in vascular tone, particularly that of the veins, forces blood toward the heart at an accelerated rate and the cardiac output increases almost exactly in proportion to the increase in mean circulatory pressure.[11]

The long-term method by which the veins control venous return and cardiac output is to increase or decrease the venous resistance.[18] It is well known, for instance, that compression of the veins by masses, either in the abdomen or the chest, increases the venous resistance, thereby decreasing the venous return and consequently the cardiac output as well. The reason for this effect can be explained as follows: The mean capillary pressure is controlled independently of the function of the heart or blood vessels by the so-called "law of the capillaries." That is, any time the capillary pressure rises too high it is readjusted within a few minutes by leakage of fluid out of the blood into the tissue spaces. Conversely, any time the capillary pressure falls too low, it is readjusted by the absorption of fluid from the tissue spaces into the blood. As a result, except for a few minutes at a time, the mean capillary pressure everywhere in the circulatory system normally remains essentially constant, so that in the normal animal the pressure gradient from the capillaries to the right atrium remains relatively invariable. The venous resistance, however, can vary tremendously. Consequently, because blood flow is equal to pressure gradient divided by resistance, the average return of blood to the heart under normal conditions is regulated to a very major extent by the resistance to blood flow in the venous system.

In summary, probably the most important single function of the veins, and yet a function that has been almost totally neglected in the past, is their ability to regulate cardiac output. Though the veins are not the only portion of the circulation to enter into the regulation of cardiac output, under normal conditions, at least, they are probably the most significant one.

Venous Pressure

Regulation of Right Atrial Pressure. Right atrial pressure is regulated by two opposing factors: first, the ability of the heart to pump blood out of the atrium, and second, the tendency for blood to flow into the atrium from the veins.[19] When the heart pumps with great effectiveness the right atrial pressure falls, but when the heart pumps very poorly the right atrial pressure rises.

On the other hand, when large quantities of blood tend to flow from the veins into the heart the right atrial pressure rises, while when the return to the heart is poor the right atrial pressure falls. Thus, the right atrial pressure is determined by a balance between the effectiveness of the heart as a pump and the tendency for blood to return to the heart.

The right atrial pressure is normally approximately 0 mm. Hg, and if it rises above this value, one can be certain that either the pumping effectiveness of the heart has become diminished or an excessively large amount of blood is attempting to return from the veins. The conditions that often diminish the pumping effectiveness of the heart include myocardial infarct, myocardial depression caused by various diseases, valvular heart disease, or other cardiac abnormalities. On the other hand, the tendency of blood to return to the heart can be greatly increased as a result of increased blood volume, excessive peripheral vascular dilatation, arteriovenous fistulae, or other effects. Sometimes, both the venous return and the pumping effectiveness of the heart are altered simultaneously. For instance, administration of epinephrine to an animal or person increases the venous return and at the same time increases the pumping effectiveness of the heart. The first of these tends to elevate the right atrial pressure, while the other tends to decrease the pressure. As a result, within very wide limits, administration of epinephrine will not greatly alter the right atrial pressure even though the cardiac output increases as much as 175 per cent.[11] Sympathetic stimulation, also, causes essentially the same effects.

Zero Reference Point for Measuring Venous Pressure. Measurements of venous pressure are an extremely sensitive index of the extent of many cardiac or vascular abnormalities. Yet these measurements must be made with great precision, for only a 1 mm. Hg change in right atrial pressure can mean as much as a 14 per cent difference in venous return. A very slight error in locating the zero point to which the right atrial pressure is referred can cause the measurements to be several mm. Hg too high or too low. Furthermore, changes in posture make a tremendous difference in the measured values. For this reason, care in using an exact zero reference point has always been emphasized by those who have used venous pressure measurements most successfully in clinical practice.[20-22]

In animal experiments, regardless of posture of the animal, the venous pressure remains essentially constant at only one single point in the circulatory system, and that is approximately at the tricuspid valve.[23] An obvious explanation for this is that an elevated pressure at this point promotes increased filling of the right ventricle, which causes the heart to pump increased quantities of blood and automatically decreases the venous pressure back toward a normal value. A depressed pressure at the tricuspid valve causes converse effects, diminished ventricular filling, accumulation of blood in the right atrium, and elevation of the pressure back toward the same normal value. This same constancy of venous pressure almost certainly occurs in the human being at the tricuspid valve. This point in the circulation, therefore, could possibly become a valuable reference point for measurement of all venous pressures, as it has already in animal experiments.

Peripheral Venous Pressure. The venous pressure in a peripheral vein is determined by many different factors operating simultaneously. These are: (1) the right atrial pressure; (2) the degree that the vein is compressed between the periphery and the right atrium; (3) the resistance of the vein; (4) the rate of blood flow through the vein; and (5) the hydrostatic pressure difference between the level of the vein and the heart.

It is quite obvious how changing the right atrial pressure affects peripheral venous pressures, for the pressure in a peripheral vein at the same level as the heart must always be at least slightly greater than that in the right atrium if blood is to continue flowing toward the heart. Also, the greater the venous resistance the greater will be the peripheral pressure, and the greater the distance of a vein below the level of the heart the greater will be the pressure because of hydrostatic pressure.

A factor that normally affects peripheral venous pressures to a very considerable extent is the compression of the veins by objects along their routes to the heart. For instance, the veins coursing through the abdomen are continually compressed by adjacent abdominal organs and even by pressure transmitted through the small amount of intra-abdominal fluid,[5] and the veins coursing through the neck and over ribs are compressed at the points of angulation. A compressed vein remains completely occluded until enough blood collects in the more peripheral portion of the vein to raise the pressure above the force of the compression. For instance, if the intra-abdominal pressure rises to 20 mm. Hg because of severe ascites, the venous pressure in the legs must rise to a value higher than 20 mm. Hg before the leg blood can flow through the abdomen toward the heart. Likewise, if a rib compresses a neck vein with a force equivalent to 5 mm. Hg, then the pressure in the vein beyond this point must rise to a value at least as high as 5 mm. Hg before the vessel will open up. Normally almost all veins entering the thorax have at least a few mm. Hg pressure applied to them from the outside. Therefore, the peripheral venous pressures in most parts of the body normally measure at least 6 to 10 mm. Hg even when the veins are at the level of the heart.

Because most veins are compressed at some point before they reach the heart, a mild in-

crease in right atrial pressure does not cause increased pressure in the peripheral veins. However, when the right atrial pressure rises to equal the force of compression against the veins, the veins become distended, and further increases in right atrial pressure then cause corresponding increases in peripheral venous pressure.[24] Thus, greatly elevated right atrial pressures are reflected by increased peripheral venous pressure, whereas increases of only a few mm. Hg usually have little effect on peripheral venous pressure. Therefore, the measurement of peripheral venous pressures is of far less diagnostic value than the measurement of right atrial pressure. If it becomes possible to make routine, easy, and accurate measurements of right atrial pressure, this will almost certainly become equally as much a part of the clinician's armamentarium as the measurement of arterial pressure. Fortunately, recent studies have shown that placing a person on his right side with his arm hanging down will cause reasonable distention of the veins in the right arm and shoulder. Therefore, venous pressures measured in the right arm under these conditions, correcting appropriately for hydrostatic pressure, are almost equal to the right atrial pressure.[25] This method of measurement deserves very serious consideration and critical evaluation by all clinicians.

Summary

The veins have long been considered to be passive conductors of blood. However, they probably have equally as many functions of importance to the circulation as the arteries. For instance, their specific characteristic of "plasticity" allows them even normally to store a large amount of blood which can be called forth by the remainder of the circulation when it is needed. And under abnormal conditions, when the blood volume becomes greatly increased, the veins can often store the extra blood without excessive elevation of the venous pressure. Also, the veins are extremely responsive to sympathetic stimulation so that during massive sympathetic discharge blood can be mobilized out of the venous reservoirs into those areas of the circulation that require extra volume.

To make blood go uphill from the dependent parts of the body, the veins have a special "venous pump" system which is so effective that the venous pressure in the feet of a normal standing person almost never rises above 10 to 15 mm. Hg.

Finally, perhaps the most important function that has yet been ascribed to the veins is their ability to regulate cardiac output, though this has been studied only to a slight extent thus far. For short periods of time, sympathetic stimulation of the veins can greatly increase or decrease the cardiac output by increasing or decreasing the mean circulatory pressure, the mean force that makes the blood flow toward the heart. Over long periods of time, the veins can regulate the cardiac output by increasing or decreasing venous resistance, probably having a more over-all effect on cardiac output than any other single factor as long as the heart itself is not incapacitated.

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